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Vivian Chit Pun, Ignatius Tak-sun Yu, Kin-fai Ho, Hong Qiu,  
Zhiwei Sun, and Linwei Tian

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# **Differential Effects of Source-Specific Particulate Matter on Emergency Hospitalizations for Ischemic Heart Disease in Hong Kong**

Vivian Chit Pun,<sup>1</sup> Ignatius Tak-sun Yu,<sup>1</sup> Kin-fai Ho,<sup>1</sup> Hong Qiu,<sup>1</sup> Zhiwei Sun,<sup>2</sup> and Linwei Tian<sup>1</sup>

<sup>1</sup>The Jockey Club School of Public Health and Primary Care, the Chinese University of Hong Kong, Hong Kong Special Administrative Region of the People's Republic of China; <sup>2</sup>School of Public Health and Family Medicine, Capital Medical University, Beijing, People's Republic of China

**Address correspondence to** Linwei Tian, 4/F, the Jockey Club School of Public Health and Primary Care, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin-NT, Hong Kong. Telephone: (+852) 2252-8879. Fax: (+852) 2606-3500. E-mail: linweit@cuhk.edu.hk

**Running title:** Effects of PM<sub>10</sub> sources on ischemic heart disease risks

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## Abstract

**Background:** Ischemic heart disease (IHD) is a major public health concern. Although many epidemiologic studies have reported evidence of adverse effects of particulate matter (PM) mass on IHD, significant knowledge gaps remain regarding the potential impacts of different PM sources. Like PM size, PM sources may influence toxicological characteristics.

**Objectives:** We identified contributing sources to PM<sub>10</sub> mass and estimated the acute effects of PM<sub>10</sub> sources on daily emergency IHD hospitalizations in Hong Kong.

**Methods:** We analyzed the concentration data of nineteen PM<sub>10</sub> chemical components measured between 2001 and 2007 by positive matrix factorization to apportion PM<sub>10</sub> mass, and used generalized additive models to estimate associations of inter-quartile range (IQR) increases in PM<sub>10</sub> exposures with IHD hospitalization for different lag periods (up to 5 days), adjusted for potential confounders.

**Results:** Eight PM<sub>10</sub> sources were identified: vehicle exhaust, soil/road dust, regional combustion, residual oil, fresh sea salt, aged sea salt, secondary nitrate and secondary sulfate. Secondary sulfate, vehicle exhaust, and secondary nitrate contributed over half of the PM<sub>10</sub> mass. While associations with IQR increases in two-day moving averages (lag<sub>01</sub>) were statistically significant for most sources based on single-source models, only PM<sub>10</sub> from vehicle exhaust (1.87%; 95% CI: 0.66, 3.10; IQR = 4.9 µg/m<sup>3</sup>), secondary nitrate (2.28%; 95% CI: 1.15, 3.42; IQR = 8.6 µg/m<sup>3</sup>) and aged sea salt (1.19%; 95% CI: 0.04, 2.36; IQR = 5.9 µg/m<sup>3</sup>) were significantly associated with IHD hospitalizations in multi-source model. Analysis using chemical components provided similar findings.

**Conclusion:** Emergency IHD hospitalization was significantly linked with PM<sub>10</sub> from vehicle exhaust, nitrate-rich secondary PM, and sea salt-related PM. Findings may help prioritize toxicological research and guide future monitoring and emission control policies.

## Introduction

Over the past decades, epidemiologic evidence has linked ambient particulate matter (PM) pollution to increased cardiovascular morbidity and mortality (Dominici et al. 2006; Peng et al. 2009). Of the cardiovascular endpoints, ischemic heart disease (IHD) is a major public health concern. IHD is defined as a narrowing of the coronary vessels that supply blood to the heart muscle. It was the leading cause of death worldwide in 2008 and the second leading cause of death in Hong Kong, with a population around 7 million and a daily average of 12 IHD deaths in 2011 (Department of Health HKSAR 2013). Evidence from the U.S. and Europe of increases in IHD events after acute exposure to elevated PM concentrations has been convincing (Dominici et al. 2006; Forastiere et al. 2005; Pope et al. 2006). It has been estimated that an average 10  $\mu\text{g}/\text{m}^3$  reduction in  $\text{PM}_{2.5}$  in 204 U.S. counties would prevent over 1,500 IHD hospitalizations per year (Dominici et al. 2006). However, previous studies in Hong Kong did not observe associations between PM and IHD hospitalizations (Wong CM et al. 2002, Wong et al. 1999) or mortality upon adjusting for gaseous pollutants (Wong TW et al. 2002). Heterogeneity in findings may reflect the fact that PM is a complex mixture of particles that vary in physical attributes, chemical composition, solubility and emission sources (Pope and Dockery 2006).

Growing research emphasis has been placed on PM sources and chemical composition (Health Effects Institute 2002; U.S. National Research Council 2004). Since PM sources generate mixtures of air pollutants with different physicochemical compositions, they might affect the relative toxicity of PM. This hypothesis is supported by toxicological evidence suggesting that PM-induced biologic effects depended on the zone of origin (e.g. industrial zone) (Alfaro-Moreno et al. 2002). Currently, the majority of studies have associated IHD, especially

myocardial infarction, with traffic-related pollution exposures estimated using surrogate pollutants (e.g., PM<sub>2.5</sub> mass, carbon monoxide, nitrogen dioxide) or direct exposure data (e.g. time spent in traffic) (D'Ippoliti et al. 2003; Lanki et al. 2006b; Peters et al. 2004). Despite these findings, it has been a challenge to quantitatively assess the impacts of multiple PM emission sources on IHD. Associations with PM sources have been inconsistent across existing studies. While some have reported associations of traffic-related and/or combustion-generated PM with increases in repolarization, inflammatory markers, and ST segment depressions among IHD patients (Lanki et al. 2006a; Yue et al. 2007), others have reported that IHD hospitalizations were not linked with traffic-related particles or other PM sources (Halonen et al. 2009; Lall et al. 2011).

In Hong Kong, although research on PM pollution and health outcomes has been active since the late-1990s, specific PM chemical components and sources responsible for the adverse effects have rarely been investigated. In this study, we took advantage of the PM<sub>10</sub> speciation data that have been available for over a decade to identify contributing sources to PM<sub>10</sub> mass using a source apportionment model, and estimate the acute effects of PM<sub>10</sub> sources on daily emergency IHD hospital admissions.

## **Methods**

### **Data**

The Hong Kong Environmental Protection Department has been collecting 24-hour filter samples of PM<sub>10</sub> regularly at six general and one roadside air quality monitoring stations since 2001 (Yuan et al. 2012). These monitoring stations were interspersed in different districts of Hong Kong. We included only data from the six general stations that are not in direct proximity

to traffic, industrial sources, buildings, or residential sources of emissions from the burning of coal, waste or oil. These stations serve to capture the air quality that the general population is exposed to on a regular basis. Twenty-six PM<sub>10</sub> chemical components were speciated from the filter samples via various analytical methods as described in detail elsewhere (Yuan et al. 2012). Speciation data between 1 January 2001 and 31 December 2007 were included in the present study. The PM<sub>10</sub> sampling frequency was on average every-sixth-day, with each station operated on a distinct sampling schedule. On a particular day, there might be none or multiple samples taken across the stations. Overall, 71% of the study days were covered by measurements from at least one station. We obtained daily mean temperature, relative humidity from the Hong Kong Observatory for the same study period.

We acquired daily counts of emergency hospital admissions between 1 January 2001 and 31 December 2007 from the Hong Kong Hospital Authority (Wong et al. 1999). Data were coded according to the 9th revision of the International Classification of Diseases (ICD-9; World Health Organization 1975). Hospitalizations for IHD (ICD-9:410-414) were extracted to construct the time series. Hospitalizations due to influenza (ICD-9:487) were extracted and treated as a potential confounder in the regression analysis.

### **Statistical analysis**

We first used the U.S. Environmental Protection Agency's positive matrix factorization (PMF) version 3.0 to identify a set of factors, interpreted as emission sources, and to estimate the source contributions to PM<sub>10</sub> mass (Hopke 2008). Station-specific measurements of elemental carbon (EC), organic matter (OM), nitrate (NO<sub>3</sub><sup>-</sup>), sulfate (SO<sub>4</sub><sup>2-</sup>), ammonium ion (NH<sub>4</sub><sup>+</sup>), chloride ion (Cl<sup>-</sup>), sodium ion (Na<sup>+</sup>), potassium ion (K<sup>+</sup>), aluminum (Al), arsenic (As), calcium (Ca),

cadmium (Cd), iron (Fe), magnesium (Mg), manganese (Mn), nickel (Ni), lead (Pb), vanadium (V) and zinc (Zn) of PM<sub>10</sub> were entered into the PMF model. Details on PMF modeling have been described elsewhere (Reff et al. 2007).

We removed the station-specific influence on the resultant concentrations of each PM<sub>10</sub> source by: 1) computing the annual mean concentration ( $X_i$ ) for each monitoring station  $i$ ; 2) subtracting the annual mean from the daily mean concentration for station  $i$  on each sample day  $j$  ( $X_{ij}$ ); 3) adding the annual mean of all stations ( $X$ ) to the resulting centered values ( $X_{ij} - X_i$ ) for each station and sampling day to produce  $X''_{ij} = X_{ij} - X_i + X$ ; and 4) taking the average of  $X''_{ij}$  over all stations (Wong et al. 2001). The final PM<sub>10</sub> sources time-series contained non-missing territory-wide mean concentrations of PM<sub>10</sub> sources for 1,805 days (71% of the 2,556 total days), which is about five days a week. All pollutant concentrations were expressed in  $\mu\text{g}/\text{m}^3$ , except for EC and OM reported in microgram of carbon in cubic meter ( $\mu\text{gC}/\text{m}^3$ ).

Generalized additive models with log link and Poisson error were used to estimate the associations between PM<sub>10</sub> sources and emergency IHD hospital admissions (Hastie and Tibshirani 1990). We adopted *a priori* model specification to guide the selection of degrees of freedom (df) for time-varying variables: smoothing splines with 8 df per year for time trend, 6 df for current day temperature and previous 3 days moving average, and 3 df for current day relative humidity and previous 3 days moving average (Bell et al. 2009; Peng et al. 2009). We included dummy variables for day of week, public holidays, and influenza epidemics (Wong CM et al. 2002).

We investigated the possible lag distribution of associations with each PM<sub>10</sub> source for exposures on the same day (lag<sub>0</sub>) and for daily exposures on the previous 1 to 5 days (lag<sub>1</sub> to lag<sub>5</sub>).



However, we focused primarily on the two-day moving average of exposure on the same day and the previous day ( $\text{lag}_{01}$ ) *a priori* based on previous studies (Wong et al. 2008; Wong CM et al. 2002). Furthermore, we conducted multi-source analysis to estimate mutually adjusted effects of  $\text{PM}_{10}$  sources on emergency IHD hospitalizations (Ostro et al. 2011). To minimize multicollinearity, we used backward elimination with an exclusion criterion of  $p > 0.10$  to select PM sources to include in the final multi-source model, while controlling for time trend, seasonality, meteorological conditions, calendar effects and influenza epidemic. Pearson's correlations were used to summarize the relationships between source-apportioned  $\text{PM}_{10}$ .  $\text{PM}_{10}$  "tracer" components, which are characterized as the typical components that are exclusively or largely derived from a particular source, were also examined, and those tracers that are found specifically in the sources included in the final multi-source model were further tested in a separate multi-pollutant model to validate the multi-source findings. A smoothing function with 3 df was applied to graphically describe the relationships between sources and IHD hospitalizations, while adjusting for time-varying confounders. For sensitivity analysis, we repeated the time-series analysis after either imputing source concentrations *for the days without samples from any stations (751 days) by linear interpolation* using the *na.approx* function in the R *zoo* package, or replacing the missing data with non-missing measurement values from the previous day. Moreover, we evaluated the impact of alternative df values (5-12) for time trend on the risk estimates. All estimates were reported as the percent increase  $[(\text{relative risk} - 1) \times 100\%]$  in daily emergency IHD hospital admissions for an inter-quartile range (IQR) increment in pollutant concentrations. Where appropriate, 95% confidence intervals were calculated. We performed all time-series analyses in the statistical environment R Software, version 2.15.0 (R Development Core Team, Vienna).

## Results

Eight PM<sub>10</sub> sources were identified, namely vehicle exhaust, soil/road dust (e.g., from exposed soil, unpaved roads), regional combustion, residual oil combustion (fuel emissions from marine vessels), fresh sea salt, aged sea salt, secondary nitrate and secondary sulfate. Figure 1 shows the estimated PM<sub>10</sub> source profiles, depicted as explained variations (EV) that indicate the relative contribution of each source to the variation of a given chemical component (Paatero and Tapper 1994). For instance, vehicle exhaust emission accounted for 80% of the variation in EC. Regional combustion emission was identified as a composite of two sources, which were wood/biomass burning based upon the abundance (i.e., large EV) of K<sup>+</sup>, and coal combustion in power plants and industrial facilities in the adjacent Pearl River Delta region based upon the abundances of As, Cd, Pb and Zn in the source profile, that cannot be further separated (Yuan et al. 2012). Table 1 summarizes the levels of PM<sub>10</sub> pollution, weather conditions, and IHD hospital admission counts. Between 2001 and 2007, the daily average concentration for PM<sub>10</sub> in Hong Kong was  $55.8 \pm 32.5 \mu\text{g}/\text{m}^3$ . Secondary sulfate accounted for the largest fraction of total PM<sub>10</sub> mass (23.6%), followed by vehicle exhaust (15.1%) and secondary nitrate (14.9%). The mean daily average temperature and relative humidity were 23.6°C and 78.3% respectively (Table 1). During the study period, there were 76,659 hospitalizations for IHD ( $30 \pm 7$  admissions per day).

Single-source models of single-day exposure lags showed similar patterns of associations for most of the PM<sub>10</sub> sources, in that IHD hospitalizations were positively associated with exposure on the same day (lag<sub>0</sub>), maximal for lag<sub>0</sub> or lag<sub>1</sub>, and lowest at later lags (lag<sub>4</sub>–lag<sub>5</sub>) (Figure 2). At lag<sub>01</sub> (Figure 3A), the source that was most strongly associated with IHD hospitalizations was secondary nitrate (2.89% increase; 95% CI: 1.83, 3.95; IQR =  $8.6 \mu\text{g}/\text{m}^3$ ), followed by vehicle

exhaust (2.35%; 95% CI: 1.24, 3.47; IQR = 4.9  $\mu\text{g}/\text{m}^3$ ) and regional combustion (2.26%; 95% CI: 0.98, 3.55; IQR = 11.7  $\mu\text{g}/\text{m}^3$ ), after adjusting for time trend, seasonality, meteorological conditions, calendar effect and influenza epidemic. Significant positive associations were also found for particles originated from soil/road dust (per 6.9  $\mu\text{g}/\text{m}^3$ ), residual oil (per 2.2  $\mu\text{g}/\text{m}^3$ ) and secondary sulfate (per 15.8  $\mu\text{g}/\text{m}^3$ ), corresponding to estimated increases in IHD hospitalizations of 0.97%–1.37%.

Correlations between source-apportioned  $\text{PM}_{10}$  were nil-to-moderate. The highest correlation coefficient was 0.67 between regional combustion and secondary sulfate, followed by that between regional combustion and secondary nitrate (0.59; Table 2). Backward elimination resulted in a multi-source regression model that included vehicle exhaust, aged sea salt, and secondary nitrate sources only (Figure 3B). All other sources, though statistically significant in single-source models, were eliminated from the final multi-source model based on  $p > 0.10$ . The estimated effects of vehicle exhaust (1.87%; 95% CI: 0.66, 3.10) and secondary nitrate (2.28%; 95% CI: 1.15, 3.42) at lag<sub>01</sub> were slightly attenuated relative to single-source model estimates, but remained significant in the final multi-source model. The association between aged sea salt and IHD hospitalizations was stronger based on the multi-source model (1.19%; 95% CI: 0.04, 2.36; IQR = 5.9  $\mu\text{g}/\text{m}^3$ ) compared with the single-source model (0.68%; 95% CI: -0.43, 1.79). Associations with "tracer" components of secondary nitrate and vehicle exhaust were similar, that is,  $\text{NO}_3^-$  (tracer for secondary nitrate) and EC (tracer for vehicle exhaust) were associated with 1.95% (95% CI: 0.68, 3.25; IQR = 3.4  $\mu\text{g}/\text{m}^3$ ) and 1.67% (95% CI: 0.58, 2.78; IQR = 1.6  $\mu\text{g}/\text{m}^3$ ) increase in IHD hospitalizations, respectively (Figure 3C).

We examined the concentration-response (C-R) relations for vehicle exhaust, aged sea salt and secondary nitrate in a multi-source model. We observed moderate positive relationships over the IQRs of source concentrations, except for aged sea salt where a neutral relationship was seen (data not shown). The risk estimates were not sensitive to alternative time-series models in which we imputed missing data (data not shown). Varying df for time trend (5-12 per year, data not shown) did not substantially change the regression results either.

## **Discussion**

Research directly delineating the health impacts of PM emission sources is relatively limited. Most studies rely on ambient concentrations of a PM chemical component as surrogate of the combined exposure to one source (Stanek et al. 2011). This *a priori* selection can be complicated when interpreting the results, because many components are emitted from numerous sources, and the same component may not serve as tracer to the same source at different locations (Sarnat et al. 2008). We joined a small but growing number of epidemiologic studies to conduct source apportionment analysis and quantitatively estimate the associations between multiple PM sources and health outcome. Early short-term air pollution studies conducted in western countries identified some associations between PM sources and mortality (Cakmak et al. 2009b; Halonen et al. 2009; Ito et al. 2006; Laden et al. 2000; Mar et al. 2000, 2006; Ostro et al. 2011), and gradually, researchers have also linked certain PM sources to hospital admissions (Andersen et al. 2007; Cakmak et al. 2009a; Halonen et al. 2009; Lall et al. 2011; Sarnat et al. 2008). Overall, these studies have reported some evidence suggesting that PM sources representing traffic/motor exhausts, regional/secondary sulfate, and coal/oil combustion may be more toxic than other PM sources, as summarized in a recent review by Stanek et al. (2011). Nonetheless, there is

insufficient evidence to draw more specific conclusions across studies. Since emission sources of air pollutants vary not only temporally, but also geographically, studies on source-apportioned PM mass under different atmospheres are needed to improve our understanding of PM-related health effects.

To our knowledge, this is the first Asian study to investigate the health impacts of multiple PM sources. We estimated the associations of short-term exposure to source-apportioned PM<sub>10</sub> mass with emergency IHD hospital admissions in Hong Kong, a coastal urban city on the boundary region of Asian continent and Pacific Ocean. In contrast to previous studies conducted in New York City and Helsinki, Finland that reported no associations of PM<sub>2.5</sub> sources with IHD hospital admissions (Halonen et al. 2009; Lall et al. 2011), we observed significant associations between IQR increases in several PM<sub>10</sub> sources and daily IHD hospitalizations for single-day lag periods up to 5 days prior. Differences between our findings and those of previous studies might be related to the longer study period (7 years), larger combined sample size in the present study, as well as the differences in pollution compositions and population susceptibility between cities. Although precise patho-physiological mechanisms connecting ambient air pollution with IHD remain to be determined, it is commonly hypothesized that PM sources may trigger and/or enhance the formation of reactive oxygen species that induce inflammation, the formation of atherosclerotic plaques as well as vaso-constriction, resulting in reduced oxygen supply of heart tissues, and thereby leading to IHD (Lawal and Araujo 2012; Peters 2011).

Secondary nitrate in PM<sub>10</sub> (per 8.6 µg/m<sup>3</sup>) was associated with the largest increases in IHD hospitalizations at lag<sub>01</sub> in both single-source and multi-source models. On the other hand, the estimated association of secondary sulfate diminished after adjusting for other sources, and

secondary sulfate was dropped from the final multi-source model based on the backward elimination criterion (data not shown). This is somewhat surprising considering that secondary sulfate accounted for the largest fraction of total PM<sub>10</sub> in Hong Kong. While most studies that examined these associations found that sulfate-rich secondary PM was more strongly associated with mortality and hospital admissions than nitrate (e.g., Ito et al. 2006; Mar et al. 2000, 2006; Sarnat et al. 2008), a few studies have reported that nitrate in PM<sub>2.5</sub>, rather than sulfate, was significant predictor of mortality (Fairley 1999; Ostro et al. 2011). Secondary nitrate and secondary sulfate derive largely from the oxidation of nitrogen oxides and sulfur dioxide emitted from combustion of fossil fuels respectively. Whilst both are acidic in nature, their strength of acidity varies greatly depending upon the city-specific interactions between local emissions, regional transports, and meteorological conditions (Schlesinger and Cassee 2003). Although animal toxicological evidence is inconclusive, it is hypothesized that acidic aerosols may lower the pH within the airways by depositing hydrogen ions, thereby triggering adverse reactions (Kelly and Fussell 2012). In China, strong economic growth and high total energy consumption have led to substantial increases in anthropogenic nitrogen and sulfur emissions over the past decades (Liu et al. 2013; Lu et al. 2010). Studies showed that emissions of nitrogen oxides and sulfur dioxide in the adjacent Pearl River Delta region due to rapid industrialization and urbanization have been the dominant contributors to secondary nitrate and secondary sulfate in Hong Kong through regional transportation (Guo et al. 2009; Yuan et al. 2012). Our finding on secondary nitrate is of particular importance for lending urgency to policy-makers, particularly in developing economies, regarding both local and regional emission control and reduction of gaseous pollutants.

We found that an IQR increment ( $4.9 \mu\text{g}/\text{m}^3$ ) in two-day moving average concentration of  $\text{PM}_{10}$  from vehicle exhaust was associated with a 1.91% estimated increase (95% CI: 0.70, 3.13%) in IHD hospital admissions after adjusting for other statistically significant sources. In accordance with these results, EC as a chemical tracer of vehicle exhaust (largely from diesel engine) was also significantly associated with IHD hospitalization risk. Vehicle exhaust-related PM are combustion-derived particles that primarily accumulate in the fine fraction of  $\text{PM}_{10}$ . Previous epidemiologic studies have reported that mobile sources  $\text{PM}_{2.5}$  and EC are stronger predictors of overall cardiovascular outcomes than other sources and components (Cakmak et al. 2009a, 2009b; Lall et al. 2011; Mar et al. 2006; Ostro et al. 2011; Sarnat et al. 2008), which is consistent with our finding of an association of IHD with vehicle exhaust PM. Plausible biological mechanisms include elevated levels of inflammatory biomarkers, impaired endothelium-dependent vasodilation, and promotion of ST-segment depression (Dales et al. 2007; Lanki et al. 2006a; Yue et al. 2007). In Hong Kong, where road density was among the highest in the world at 254 vehicles per kilometer of road in 2009, exposure to traffic-related air pollution is ubiquitous (World Bank 2012). These findings on vehicle exhaust particles stress the importance of continuous reduction of overall traffic and related emissions, and configuration of urban environment to reduce personal exposure to traffic.

We observed that aged sea salt was associated with an increased risk of IHD hospitalizations after adjusting for other sources. Sea salts are most abundantly found in the coarser fraction of  $\text{PM}_{10}$ . While Mar et al. (2006) reported that sea salt was consistently associated with elevated cardiovascular and total mortality in Atlanta, Georgia across the various source apportionment analyses in an inter-method comparison study, most epidemiologic studies that estimated this

association found no relationship between sea salt and health outcomes (Andersen et al. 2007; Gent et al. 2009; Lanki et al. 2006a; Ostro et al. 2011). Several studies suggested possible relations between sea salt and secondary nitrate, as nitric acid may react with marine particles to form coarse mode nitrate along coastal areas (Lee et al. 2008; Zhuang et al. 1999). However, we observed null-to-weak correlations between secondary nitrate and sea salts. Such association should be investigated further.

Our findings add to the existing literature in several ways. First, we examined air pollution association with specific cardiovascular endpoint, as opposed to a broad composite endpoint of different cardiovascular events, to provide better insight into the plausible biologic mechanisms. Second, with nearly 80,000 IHD hospital admissions over 7 years, our study was well powered to detect statistically significant associations. Moreover, this was one of the few epidemiologic studies that focused on exposure to source-apportioned PM<sub>10</sub>, while most existing studies were based on source-apportioned PM<sub>2.5</sub>. This allowed us to identify adverse associations of not only sources that primarily generate finer mode PM<sub>10</sub>, but also those that produce coarser mode PM<sub>10</sub>.

Although we provided evidence of the health impacts of several PM<sub>10</sub> sources in Hong Kong, these findings should be interpreted with caution. While backward elimination procedure was used to identify a subset of predictors with the most statistically significant relationship with IHD hospitalization, this approach might not guarantee a truly "best" reduced model (Breiman 1996). The importance of PM<sub>10</sub> sources (e.g., regional combustion, secondary sulfate) excluded from the final multi-source model should not be diminished, as the statistical elimination procedure does not indicate or account for biological importance. Another limitation of this study was the every-sixth-day sampling scheme for the PM<sub>10</sub> speciation data, resulting in nearly one-third of



study days without samples from any stations. Exposure misclassification error might exist, however, our risk estimates were insensitive to alternative interpolation methods (data not shown). Moreover, PM from local emissions (e.g., vehicle exhaust, soil/road dusts) tends to have more error than regional PM sources (e.g., secondary PM), given their higher spatial heterogeneity (Ito et al. 2004). Such issues of representativeness associated with PM sources may hinder the interpretations of the relative strengths of the observed associations in monitor-based studies of ambient PM pollution.

## **Conclusion**

We report evidence that PM<sub>10</sub> from vehicle exhaust, nitrate-rich secondary PM, and sea salt-related PM were significantly associated with elevated IHD hospitalization risks in Hong Kong. This study joins a growing body of literature to report evidence of adverse effects of source-apportioned PM mass, which would help prioritize research on the biologic mechanisms linking PM pollution to cardiac events and guide future monitoring and emission control policies.

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**Table 1.** Descriptive statistics for PM<sub>10</sub> sources, meteorological factors and number of emergency hospital admissions in Hong Kong, 2001-2007.

<b>Variables</b>	<b>No. of days</b>	<b>Daily mean <math>\pm</math> SD</b>	<b>Proportion (%)</b>	<b>IQR</b>
<b>Emergency hospital admissions (counts)</b>				
Ischemic heart diseases (IHD)	2556	30 $\pm$ 7		9
<b>Meteorological conditions</b>				
Temperature (°C)	2556	23.6 $\pm$ 4.9		8.1
Relative humidity (%)	2556	78.3 $\pm$ 9.9		11.4
<b>PM<sub>10</sub> concentration (<math>\mu\text{g}/\text{m}^3</math>)</b>				
Total PM <sub>10</sub>	1805	55.8 $\pm$ 32.5	100	44.8
Vehicle exhaust	1805	8.4 $\pm$ 3.7	15.1	4.9
Soil/road dust	1805	7.5 $\pm$ 9.0	13.4	6.9
Regional combustion	1805	7.5 $\pm$ 9.3	13.5	11.7
Residual oil	1805	2.4 $\pm$ 2.5	4.3	2.2
Fresh sea salt	1805	2.1 $\pm$ 2.7	3.7	2.0
Aged sea salt	1805	7.2 $\pm$ 4.4	12.8	5.9
Secondary nitrate	1805	8.3 $\pm$ 8.8	14.9	8.6
Secondary sulfate	1805	13.2 $\pm$ 12.7	23.6	15.8

Abbreviations: SD, standard deviation; IQR, inter-quartile range.

**Table 2.** Pearson's correlation among the estimated sources of PM<sub>10</sub>.

	<b>PM<sub>10</sub></b>	<b>Vehicle exhaust</b>	<b>Soil/road dust</b>	<b>Regional combustion</b>	<b>Residual oil</b>	<b>Fresh sea salt</b>	<b>Aged sea salt</b>	<b>Secondary nitrate</b>
PM <sub>10</sub>	1							
Vehicle exhaust	0.48	1						
Soil/road dust	0.58	0.21	1					
Regional combustion	0.84	0.49	0.38	1				
Residual oil	0.40	0.35	-0.02	0.29	1			
Fresh sea salt	0.10	-0.18	0.12	-0.07	-0.11	1		
Aged sea salt	0.07	-0.27	0.08	-0.22	-0.11	0.23	1	
Secondary nitrate	0.76	0.34	0.29	0.59	0.34	0.24	0.05	1
Secondary sulfate	0.78	0.30	0.20	0.67	0.38	-0.15	-0.02	0.43



## Figure Legends

**Figure 1.** PM<sub>10</sub> source profiles, indicated by explained variations (EV) that estimate how much a source explains the variation of a particular chemical component.

**Figure 2.** Percent change (95% CIs) in emergency IHD hospital admissions per IQR increment in PM<sub>10</sub> mass and sources at different lag periods, adjusted for meteorological factors, seasonal and temporal trend, day-of-week and influenza epidemic. See Table 1 for individual IQR values.

**Figure 3.** Percent change (95% CIs) in emergency IHD hospital admissions per IQR increment in two-day moving average concentration ( $\text{lag}_{01}$ ) of PM<sub>10</sub> sources based on single-source models (A), multi-source model (B), and PM<sub>10</sub> trace elements based on multi-pollutant model (C). All models were adjusted for time trend, seasonality, meteorological conditions, calendar effects and influenza epidemic. See Table 1 for individual IQR values for sources, and the IQR for EC (tracer for vehicle exhaust), Na<sup>+</sup> (for aged sea salt), and NO<sub>3</sub><sup>-</sup> (for secondary nitrate) was 1.6, 1.2 and 3.4 µg/m<sup>3</sup> respectively.

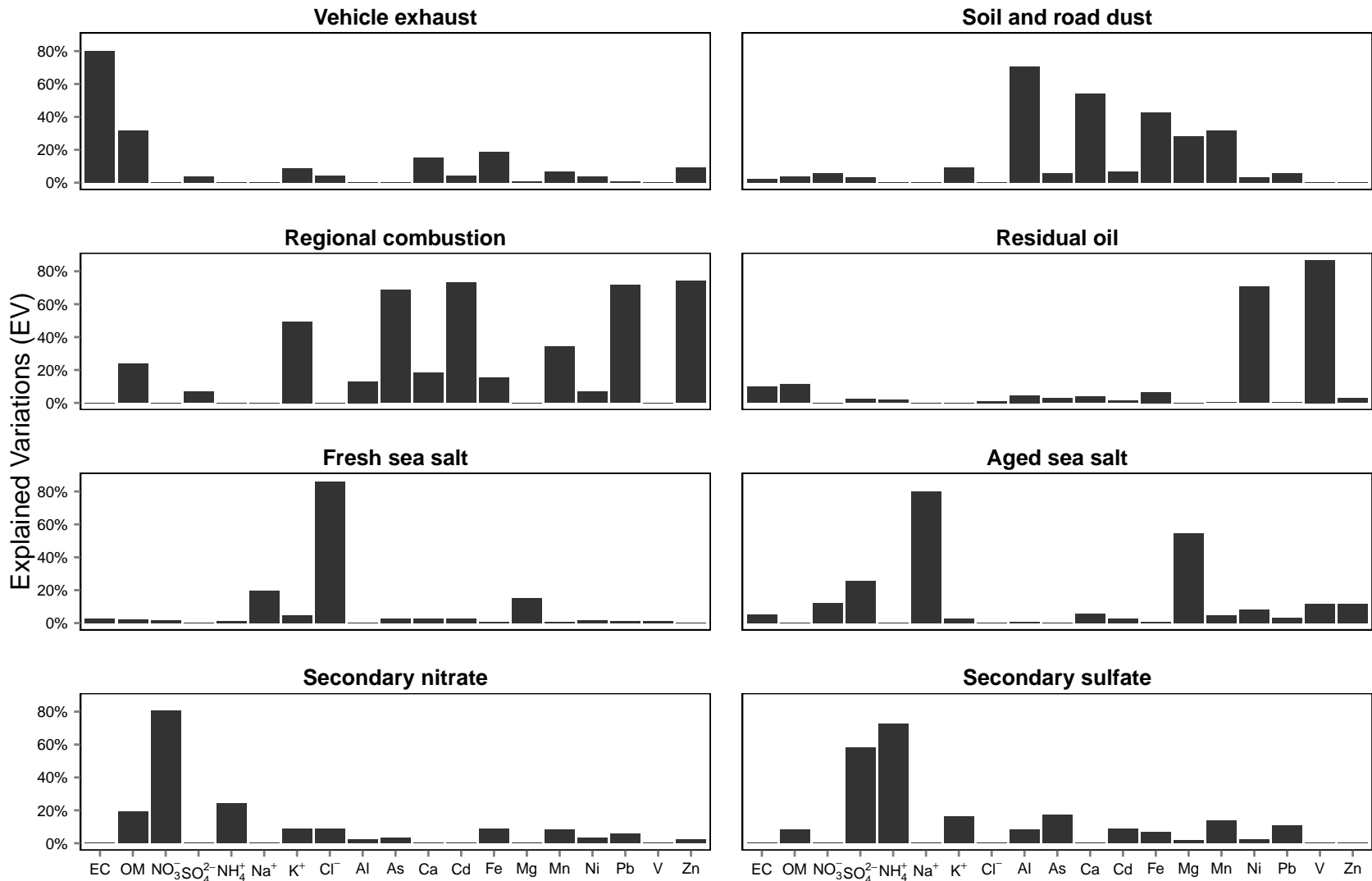


Figure 1.

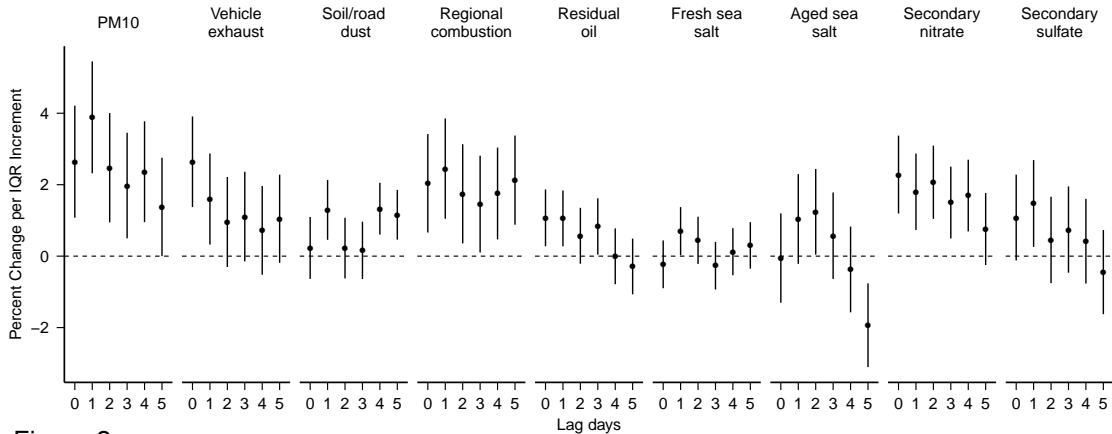


Figure 2

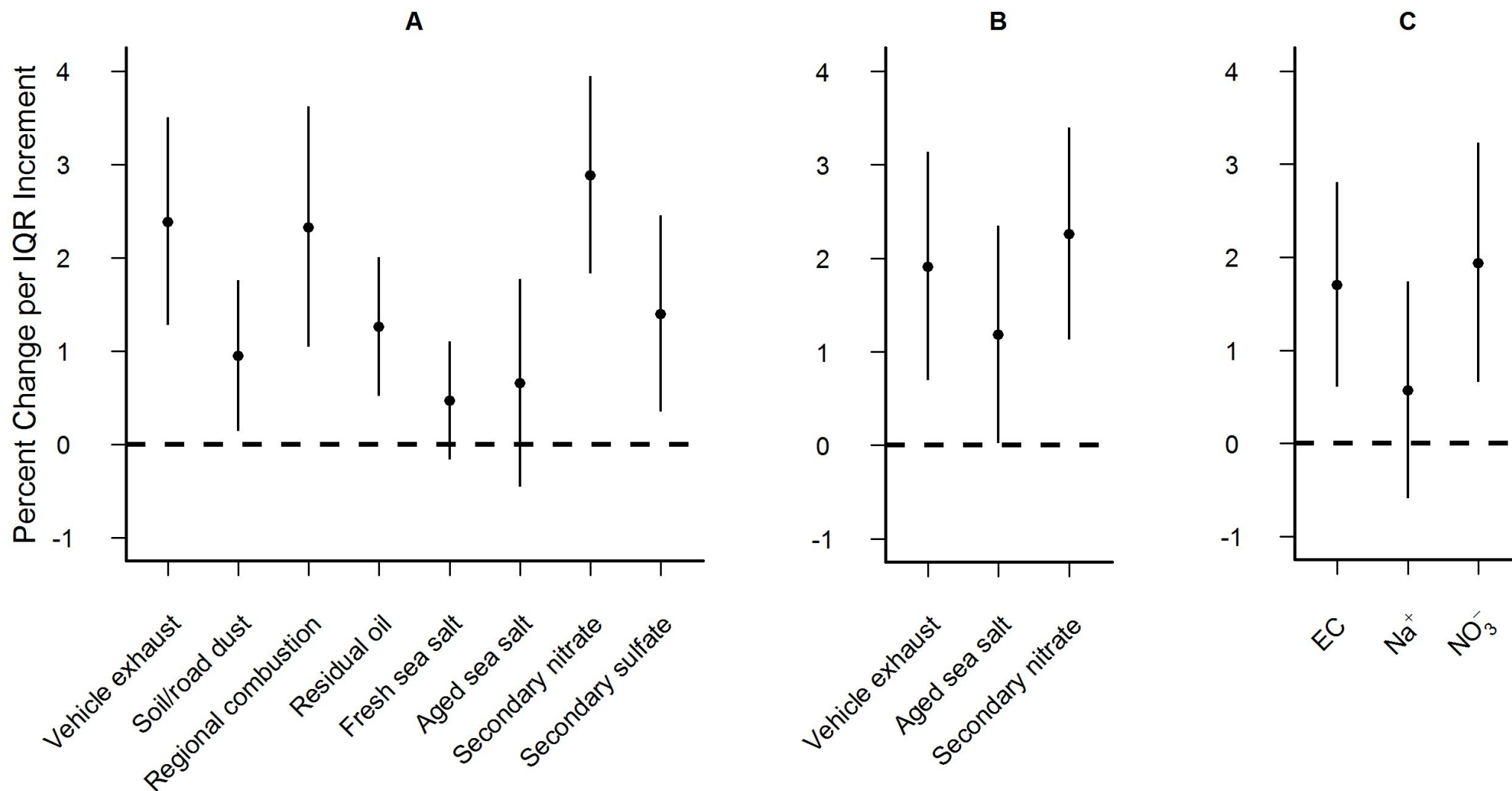


Figure 3